Tobacco Smoke

Cotinine

CAS No. 486-56-6

General Information

Tobacco use is the most important, preventable cause of premature morbidity and mortality in the United States. The consequences of smoking and the use of smokeless tobacco products are well known and include an increased risk for cancer, emphysema, and cardiovascular disease. For example, lung cancer is the leading cancer-related killer of both men and women in the United States, and smoking is by far the leading cause of lung cancer.

Environmental tobacco smoke (ETS) is a known human carcinogen, and persistent exposure to ETS is associated with an increased risk for lung cancer and other diseases. Children are at particular risk from ETS, which may exacerbate asthma among susceptible children and greatly increase the risk for lower respiratory-tract illness, such as bronchitis and pneumonia, among young children.

Cotinine is a major metabolite of nicotine and is currently regarded as the best biomarker in active smokers and in nonsmokers exposed to ETS. Measuring cotinine is preferred over measuring nicotine because cotinine persists longer in the body. Cotinine can be measured in serum, urine, saliva, and hair. Nonsmokers exposed to typical levels of ETS have cotinine levels of less than 1

Table 60. Cotinine

Geometric mean and selected percentiles of serum concentrations (in ng/mL) for the non-smoking U.S. population aged 3 years and older, National Health and Nutrition Examination Survey, 1999-2000.

	Geometric mean (95% conf. interval)	Selected percentiles (95% confidence interval)						Sample size
		10th	25th	50th	75th	90th	95th	
Total, age 3 and older	*	< LOD	< LOD	.059 (<LOD-.070)	.236 (.180-.310)	1.02 (.740-1.27)	1.96 (1.64-2.56)	5999
Age group								
3-11 years	*	< LOD	< LOD	.109 (.064-.180)	.500 (.290-1.02)	1.88 (1.19-3.09)	3.37 (1.79-4.23)	1174
12-19 years	*	< LOD	< LOD	.107 (.080-.163)	.540 (.371-.762)	1.65 (1.25-2.11)	2.56 (2.35-3.23)	1773
20 years and older	*	< LOD	< LOD	< LOD	.167 (.137-.200)	.630 (.520-.863)	1.48 (1.23-1.77)	3052
Gender								
Males	*	< LOD	< LOD	.080 (.060-.100)	.302 (.220-.390)	1.20 (.890-1.56)	2.39 (1.78-3.06)	2789
Females	*	< LOD	< LOD	< LOD	.179 (.135-.250)	.850 (.590-1.14)	1.85 (1.41-2.37)	3210
Race/ethnicity								
Mexican Americans	*	< LOD	< LOD	< LOD	.139 (.107-.182)	.506 (.340-.813)	1.21 (.813-1.84)	2242
Non-Hispanic blacks	*	< LOD	< LOD	.131 (.110-.150)	.505 (.400-.625)	1.43 (1.22-1.66)	2.34 (1.89-2.97)	1333
Non-Hispanic whites	*	< LOD	< LOD	.050 (<LOD-.070)	.210 (.150-.313)	.950 (.621-1.40)	1.92 (1.54-2.74)	1949

< LOD means less than the limit of detection, which is 0.05 ng/mL.

* Not calculated. Proportion of results below limit of detection was too high to provide a valid result.

ng/mL, with heavy exposure to ETS producing levels in the 1-10 ng/mL range. Active smokers almost always have levels higher than 10 ng/mL and sometimes higher than 500 ng/mL.

Interpreting Serum Cotinine Levels Reported in the Table

Table 60 presents data for the U.S. nonsmoking population aged 3 years and older. For these results, nonsmoking is defined as a serum cotinine level less than or equal to 10 ng/mL. Choosing a cutoff of 15 ng/mL makes little difference in the results. The LOD for these measurements was 0.050 ng/mL.

From 1988 through 1991, as part of NHANES III, CDC determined that the median level (50^{th} percentile) of cotinine among nonsmokers in the United States was 0.20 ng/mL (Pirkle et al., 1996). Table 60 shows that the median cotinine level in 1999-2000 has decreased to 0.059 ng/mL—more than a 70% decrease. This reduction in cotinine levels suggests a dramatic reduction in exposure of the general U.S. population to ETS since the period 1988-1991. Compared with results for the period 1988-1991 for population groups defined by age, gender, and race/ethnicity (Pirkle et al., 1996), cotinine levels declined in all categories.

Covariate-adjusted geometric means were not calculated because more than 40% of the population had cotinine levels less than the LOD. At comparable percentiles, men have higher cotinine levels than women, and non-Hispanic blacks have higher levels than non-Hispanic whites or Mexican Americans. Higher levels of cotinine have been reported for non-Hispanic blacks (Caraballo et al., 1998). As seen previously (Pirkle et al., 1996), males continue to have higher levels than females, and people aged 20 years and older have lower levels than those younger than 20 years of age.